male-killing strain described in Upolu and Savii [15]. Populations infected with this second strain can resist invasion from the male-killer *w*Bol1, adding more complexity and yet another level of evolutionary conflict to the mix.

Compelling support for rapid evolution often stems from laboratory studies, yet here the research documents transitions in the wild without experimental interference. Charlat *et al.* [2] have caught evolution-in-action on islands that have really lived up to their 'natural laboratory' tag. Evidence indicating rapid host counter-adaptation after 10 generations, obtained without sustained targeted selection possible in experimental evolution studies, is impressive and shows what an effective agent of punctuated change conflict-based evolution can be. The data captured from the natural environment avoid criticisms of artificial selection, commonly levelled at lab experiments. Powerful evidence from field systems is understandably rare, because it requires being around at the right place, at the right time, *and* measuring the right trait(s) using the right tools. The issue of timing is particularly keen here, as the extreme female-biased sex-ratio seems to have persisted for at least 78 years — between 1923 and 2001 — before rapidly switching and approaching parity in 2005/2006 [2,7]. Dynamics over this period, and in the future, beg many further questions of this powerful system. It would be extremely informative to know how the sex ratio varied in the female-biased population before the suppressor joined the fray? How did the suppressor enter the population: via immigration or mutation? What are the exact mechanisms by which *w*Bol1 kills male embryos, and how does the suppressor nullify this effect? What are the costs, if any, of carrying the suppressor? When, if ever, will (*w*Bol1) change and out-evolve the suppressor? The continued effective combination of population and behavioural ecology with modern molecular genetic techniques, as well as some laboratory experimental control, should allow some of these important evolutionary questions to be answered.

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Centre for Ecology, Evolution and Conservation, School of Biological Sciences, University of East Anglia, Norwich Research Park, Norwich NR4 7TJ, UK.
E-mail: o.martin@uea.ac.uk, m.gage@uea.ac.uk

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Autism: Not Interested or Not 'Tuned-in'?

Recent studies of perceptual adaptation to faces have revolutionised our understanding of neural mechanisms that support face recognition. A new study has applied this approach to autistic spectrum disorders, revealing severe deficits in such adaptation.

Greg Davis and Kate Plaisted

Human faces differ subtly along many different dimensions, yet the human brain is able to distinguish between them with a rapidity and precision unmatched by any artificial recognition system. Given their central role in human interaction, the brain's face-processing mechanisms have understandably been the subject of intense scrutiny, and functional imaging (fMRI) studies have located such mechanisms are present in regions of occipitotemporal cortex. However, progress in understanding *how* these mechanisms function has been frustratingly slow. Some theories suggest that the brain encodes many individual faces independently of each other, whereas other theories postulate that all faces are coded relative to an average or 'prototype' face. Indeed, it is only recently that simple, but ingenious studies of the way that face processing mechanisms alter their responses ('adapt') when exposed to new face

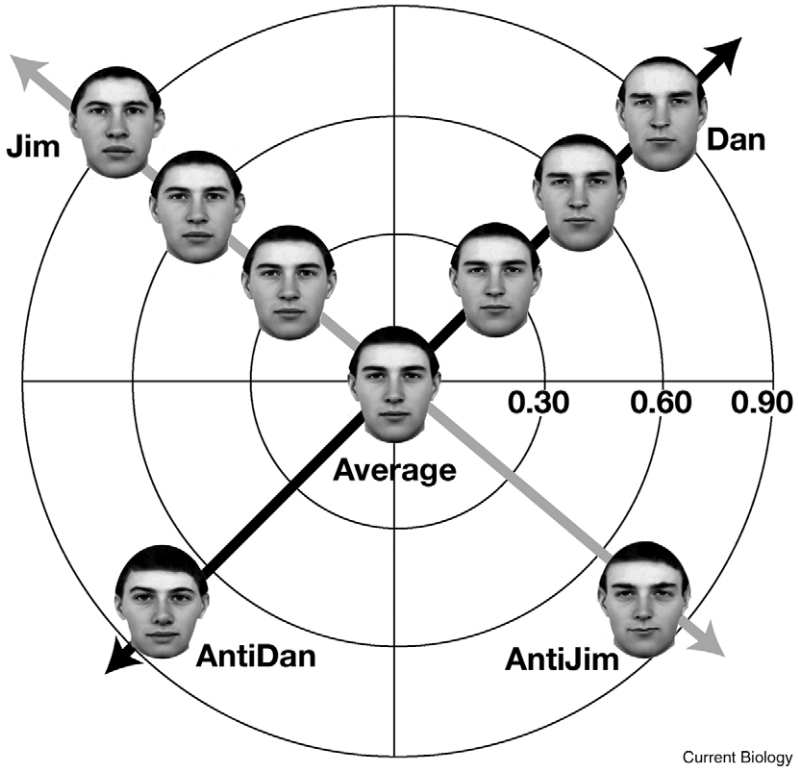

Figure 1. Schematic illustration showing the two-dimensions along which the face stimuli they employed varied.

Photo from Pellicano *et al.* [4]; see text for further details.

stimuli have begun to resolve this enduring controversy [1].

To illustrate the logic of these studies, consider a limited set of faces that differ in a systematic way along two arbitrary dimensions (schematised in Figure 1). The first of these is the degree to which a face shares the properties of the face with identity 'Dan' ('Proportion Dan'), ranging from Dan himself (top-right of the figure) to faces that are the polar opposite of Dan's face in physical terms ('Anti-Dan': see bottom-left of the figure). The other, orthogonal dimension ranges from 'Jim' to 'Anti-Jim', and the average of the set of faces lies mid-way between Dan and Anti-Dan, and mid-way between Jim and Anti-Jim. In this diagram, therefore, faces can be encoded in terms of their position along two dimensions relative to the average or 'prototypical' face. Such a diagram is, of course, of little interest in and of itself; however, as we discuss next, studies of adaptation have provided evidence that the human brain encodes faces in a logically similar, though much more complex, manner.

Typically, when one adapts (is exposed) to a stimulus towards one end of any perceptual continuum, there is a 'negative after-effect' whereby subsequent stimuli appear shifted toward the other end of that continuum. These effects were thought to arise only for simple mechanisms at early stages of visual processing; however, a burgeoning literature now indicates that some such effects reflect specific adaptation of high-level face processing mechanisms [2,3]. If an observer, having been familiarised with the faces in our example above, were to adapt to anti-Jim for several seconds, they would then identify the prototype as more 'Jim-like' — shifted toward the Jim end of the Jim/Anti-Jim continuum. Such effects show that our perception of one face is not independent of our adaptation to others. Moreover, the after-effects are largest when the adapting face and the subsequent face lie directly on opposite sides of the prototype, highlighting the special status of the prototype face. These findings reveal basic aspects of face encoding in the human brain that would normally be expected to arise for all human observers. It is for this reason that the findings of a new study by Pellicano *et al.* [4], published recently in *Current Biology*, are particularly remarkable. This study has demonstrated that in individuals with autism adaptation to faces is significantly attenuated.

Pellicano *et al.* [4] compared face after-effects in typical children to those in children with autism. Children were first trained to identify the two target faces ('Jim' and 'Dan') along with faces that were a particular proportion Dan or Jim. These latter faces could be 40% Jim, 60% Jim, 40% Dan or 60% Dan. As the proportion of Dan in a face increases, the children should judge it to be Dan more often; similarly, as the proportion 'Jim' increases, the face should be judged as Jim more often. Such was the case for both groups of children. However, the children then adapted to 80% 'Anti-Dan' or 80% 'Anti-Jim', which, in typical adults, would cause subsequent faces to appear more 'Dan' and more 'Jim', respectively (due to the negative after-effect described above). Whilst the after-effect was observed in typical children, it was substantially reduced in children with autism compared to the typically-developing group.

The implications of these findings are too complex to discuss comprehensively here, as autism is a multifaceted disorder characterised by impairments in social communication and language, and also typified by repetitive behaviours. However, a core problem common to all individuals with the disorder is poor decoding of others' intentions from their behaviour during social interactions. Two distinct, though not mutually exclusive, types of explanation have been posited to explain this deficit. First, it may be that individuals with autism *choose* not to attend to faces and other socially relevant stimuli in day-to-day life; indeed, a tendency in the disorder has been

documented when viewing video sequences of social interactions in an unconstrained manner [5]. Alternatively, however, such difficulties in autism might reflect more fundamental problems in *perceiving* social stimuli, including faces and voices [6]; even under more constrained viewing conditions, individuals with autism seem to show poorer identity and emotion recognition [7].

Pellicano *et al.*'s [4] study provides clear evidence of poor social perception, revealing that adaptation of face mechanisms is severely abnormal in autism. The children with autism in their study were clearly attending to the faces as their performance in baseline conditions was similar to that of the typical children, yet fundamental differences in face-processing were evident. It is not simply the case, therefore, that children with autism were uninterested in the stimuli that Pellicano *et al.* used. Rather, it seems that their face-recognition mechanisms were not adapting to the new stimuli. The function of adaptation, in other domains of visual processing, seems to be to provide stability of visual categorisation, by 're-tuning' perceptual mechanisms to take account of prevailing conditions (for example, ensuring that a grey object will look grey even when appearing under coloured illuminants). If this does not operate for face perception in autism, categorisation of faces may be unstable from one day to the next, even though the individual can in each case distinguish one stimulus from another.

Two final points also merit particular discussion. First, an important question for future research to address is whether face adaptation deficits have substantial consequences for social and communicative processing in general. Suggestive evidence that they do is already apparent from Pellicano *et al.*'s [4] finding that the severity of autistic symptoms in each of their individuals with autism showed a clear relationship to their deficit in adaptation. Second, the work also demonstrates neatly the value of studying the interplay between functional neural subcomponents in disorders, rather than between brain regions. fMRI studies suggest that when attention to faces is maintained, children with autism recruit the same neural areas when viewing faces that typical children do, suggesting 'normal' face processing. Instead, the trick to understanding face processing abnormalities in autism may not simply lie in gross activation or anatomical differences, but rather in the substantial, if subtle, interactions between representations sharing the same neural underpinnings.

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Department of Experimental Psychology, University of Cambridge, Downing Street, Cambridge CB2 3EB, UK.
E-mail: gjd1000@cam.ac.uk, kcp1000@cam.ac.uk

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Sexual Selection: Signals to Die for

Sexual signals are conspicuous and are typically assumed to be energetically costly, which keeps them honest. A recent study on fireflies has found that signal production is energetically cheap, but signalling remains expensive because of eavesdropping predators.

Fleur E. Champion de Crespigny and David J. Hosken

The sexual signals that males use to serenade potential mates are typically extravagant and conspicuous [1]. These signal characteristics are great because they enhance reproductive success, for example by making signallers easy to find. But they also make signals costly if they deplete the energy reserves needed for reproduction, or if they alert predators to the presence of a snack in the form of the signaller. Signal costs such as these are important because they ensure signals are honest indicators of mate quality [1,2]. That is, only high quality males are able to produce the costly signals females pay attention to, much as Rolls Royce motorcars and private jets are honest signals of human wealth. These costs also act as a brake on signal exaggeration, and energetic and predation costs have been widely documented. However, multiple signal costs are rarely investigated in one species so we only have a rudimentary understanding of the relative importance of different costs in signal evolution. A new study by Woods *et al.* [3] has partly redressed this gap in our understanding by quantifying the energetic and predation costs of bioluminescent signals in *Photinus* fireflies. Their results contrast somewhat with findings in other groups, but it is not entirely clear why.

One of the few instances where the multiple costs of a sexual signal